EXHIBIT O

North Shore-LIJ Health System is now **Northwell Health**

Occupational Medicine, Epidemiology and Prevention

May 3, 2016

Kevin Paul, Esq.
Simon, Greenstone, Panatier, Bartlett
3232 McKinney Avenue
Suite 610
Dallas, Texas 75204

Re: Doris Jackson

Dear Mr. Paul:

I am writing to report the results of my evaluation of the materials listed below pertaining to Ms. Doris Jackson. I have reviewed these materials in the context of my pre-existing knowledge, training, and experience in the field of occupational medicine. These materials are of the type I and other specialists in occupational medicine normally rely upon and are sufficient to form a reliable basis for my opinions contained within this report. All of the opinions stated in this report are given within a reasonable degree of medical certainty.

This report and the opinions stated in the report are based on the listed materials and my 24 years of training, education, and experience in the area of asbestos-related occupational medicine. Over the past 24 plus years, I have had the opportunity to evaluate and treat hundreds of patients with asbestos exposure, many of whom have asbestos related diseases.

Qualifications:

I am a physician licensed in the State of New York, specializing in the field of occupational and environmental disease. I have been a practicing physician since I graduated from medical school in 1988. A copy of my Curriculum Vitae, dated April 15, 2016 is attached to the report as Exhibit 1.

I attended the University of Chicago and received a Bachelor of Arts degree with Honors, with a major of History, Philosophy and Social Studies of Science and Medicine. I then continued at the University of Chicago – Pritzker School of Medicine, where I obtained my medical degree in 1988. I was elected to the Alpha Omega Alpha Honor Society, and was also awarded an American Medical Women's Association Award. Following medical school graduation, I was an intern and resident in Internal Medicine at Yale University – Yale New Haven Hospital from 1988 – 1991. Upon completion of my Internal Medicine Residency program, I completed a second residency at the Mount Sinai School of Medicine in Occupational Medicine, from 1991 – 1993. During my Occupational Medicine Residency Program, I obtained my Master of Science Degree in Community Medicine (equivalent degree to a Masters of Public Health) in 1993. I began to evaluate dozens of patients with asbestos exposure during my residency program at Mount Sinai. I am board certified in Occupational Medicine and in Internal Medicine. I have become recertified in Internal Medicine two times.

Following completion of my residency training in Occupational Medicine, I was awarded a Fellowship in Occupational Medicine from the Foundation for Occupational Health and Research. I continued at Mount Sinai, where I joined the faculty, and continued to evaluate patients with asbestos exposure. I became Vice Chair of the Department of Preventive Medicine in 2001. I was Director of the New York/New Jersey Education and Research Center from 2006 – 2010, and had been Director of the Residency Program in Occupational Medicine from 1998-2006. I was also the Director of the Mount Sinai World Trade Center Medical Monitoring and Treatment Program from 2006 – 2010, although my involvement with the World Trade Center medical programs started in 2001, when I began to evaluate patients with exposure to the World Trade Center disaster, and was initially Medical Core Director of the World Trade Center Worker and Volunteer Medical Screening Program (2002-2004), and Co-Director of the World Trade Center Medical Monitoring and Treatment Program (2004-2006). I have published over fifty articles in the peer-reviewed literature.

In 2010 I left the Mount Sinai School of Medicine to become the Founding Chair of the Department of Population Health at Northwell Health and Hofstra Northwell School of Medicine (formerly known as North Shore University Health System). The Department changed its name in 2014 to Occupational Medicine, Epidemiology and Prevention.

I have evaluated hundreds of patients with asbestos exposure in my career in occupational medicine, spanning nearly 25 years. I currently direct the Occupational and Environmental Medicine Center of Long Island, providing occupational health services to

patients in the metropolitan New York area. Over the past year alone, I have supervised the examination of or directly examined nearly 500 patients with asbestos exposure, as we have greatly expanded our clinical services. Over the course of the past 25 years, I have evaluated dozens of patients with malignant mesothelioma and lung cancer due to asbestos exposure. I have kept abreast of the scientific and medical literature regarding the diagnosis and causation of mesothelioma. I have personally evaluated cases of mesothelioma where the exposure was brief, and have also seen cases of mesothelioma in individuals whose only exposure to asbestos was from family members who worked with asbestos and brought their asbestos contaminated clothes home.

I have testified as an expert on the health effects of asbestos, providing both deposition and trial testimony. I have attached as Exhibit B a list of my deposition and trial testimony for the past four years.

My hourly rate of compensation for medical and document review and expert report generation is $500/hour. My hourly rate for deposition and trial testimony is also $500/hour.

Materials Reviewed:

I have had the opportunity to review the medical records and deposition transcripts of Ms. Jackson. I was provided with the following information:

1. Medical records of Doris Jackson
2. Deposition testimony of Doris Jackson 09/28/15 and 09/29/15 with exhibits
3. Exposure History Sheet
4. Death Certificate, Issued by The District of Columbia, Date of Death, October 28, 2015
5. Discovery answers served in the case
6. Expert Report, Mr. Sean Fitzgerald, May 1, 2016
7. Letter and final analytical results from S. Lewin to A. Weissler dated August 3, 1972
8. Memorandum from A. Weissler to R. Shaffner dated July 31, 1973
9. Cosmetic Toiletry and Fragrance Association documents
10. Company product testing documents produced in litigation
11. Proceedings of the Symposium on Talc, Washington DC by A. Langer, May 8, 1973

Ms. Jackson's Medical and Exposure History:

Ms. Jackson is an 84 year old woman who developed shortness of breath with exertion in September 2014, along with a cough and chest tightness. The shortness of breath worsened and Ms. Jackson went to Washington Hospital Center on November 3, 2014, where she was subsequently admitted. She had shortness of breath, a cough and

chest discomfort. A chest x-ray showed a very large left pleural effusion. Ms. Jackson had a thoracentesis on November 4, 2014, with approximately 1,500 cubic centimeters of fluid removed from the left chest. Her pulmonary symptoms improved following the fluid removal. The cytology showed carcinoma cells, with carcinoma cells also present in the cell block. No evidence of lymphoma or leukemia was noted on flow cytometry. Dr. Sharma, an oncologist, saw Ms. Jackson in the hospital after the thoracentesis cytology showed malignant cells. A CT angiogram was done on November 4, 2014 and showed only a large left pleural effusion with compressive atelectasis of the left middle and lower lobes. No pulmonary embolism was seen. There were ground glass opacities in the left upper lobe and right lung, suggestive of pulmonary edema, and less likely, alveolitis. An abdominal CT scan on November 6, 2014 showed a partially imaged large left pleural effusion with enhancing soft tissue along the medial pleura, suspicious for metastatic disease with a malignant pleural effusion. There was a right adrenal mass noted and a mixed solid/cystic left renal lower pole mass, and renal cell carcinoma should be considered. Gallstones were also noted. There was a large lipoma in the pelvis. A bone scan was negative for metastatic lesions but showed osteoarthritis. Dr. Christopher Eger, a thoracic surgeon, saw Ms. Jackson in the hospital. He recommended a thoracoscopy with fluid management with a pleurodesis and possibly a chest tube. Ms. Jackson wished to return the following week for surgery so that she could attend a funeral.

Ms. Jackson underwent a thoracoscopy on November 13, 2014 at Washington Hospital Center. At the time of surgery, Dr. Eber removed around 2,300 cubic centimeters of serosanguinous fluid and noted bulky posterior parietal pleural implants, which he biopsied. He then insufflated talc and inserted a chest tube. Additional talc was administered through the chest tube on November 15, 2014. The chest tube was removed on November 17, 2014, and she was discharged home later on November 17, 2014.

The pathology showed a malignant epithelial neoplasm, consistent with metastatic poorly differentiated carcinoma. The pathology was reviewed at UCLA Medical Center, and the pathology was felt to be malignant mesothelioma, biphasic type with 90% epithelioid subtype and focal sarcomatoid features. The staining patterns showed focal positivity for RCC and was positive for CA9.

Ms. Jackson had a mammogram on November 20, 2014 that showed no mammographic evidence of malignancy. Ms. Jackson went to see Dr. Frederick Smith, a medical oncologist, on November 20, 2014. He ordered a PET/CT scan A PET/CT scan on November 24, 2014 showed a moderate, partially loculated left pleural effusion. There were multifocal regions of increasing FDG utilization within the pleural fluid, including some areas admixed with regions of higher density on CT which suggested pleurodesis-related findings, although malignancy could also account for the findings. NO definite extrathoracic disease was seen. There was a right adrenal low-density nodule that appeared to be an adenoma. A complex appearing left mid to lower pole renal lesion was seen, which appeared to be a cyst and a possible second nodule that could represent a renal cell carcinoma. Ms. Jackson started chemotherapy with Carboplatin and Alimta while she was in Maryland. She then moved to California and continued chemotherapy with Dr. Olevsky, and received her second cycle of chemotherapy at UCLA. [Records of

her chemotherapy were not available for my review; the records note that she received four cycles of neo-adjuvant chemotherapy with Alimta and Carboplatin.]

Dr. Robert Cameron, a thoracic surgeon, saw Ms. Jackson on February 4, 2015. In her intake form, she noted that she was exposed for over thirty years to ceiling pipes with degrading insulation while working as a teacher in the DC Public Schools. An MRI of the abdomen was done on February 5, 2015. There was a mass in the right adrenal gland. There was a non-enhancing lesion projecting from the lower pole of the kidney consistent with a simple cyst with a small focus of either hemorrhage or proteinaceous debris. A PET/CT scan on February 11, 2015 showed a small pericardial effusion. There was no lymph node enlargement. There was a stable right upper lobe ground glass nodule and right lower lobe scarring versus atelectasis. There was left greater than right biapical pleural parenchymal scarring. Dr. Karim Chamie, a urologist, saw Ms. Jackson on February 12, 2015 because of the pathology staining. Dr. Chamie reviewed the films and felt that Ms. Jackson had a benign left renal cyst and likely mesothelioma of non-renal origin. There was no lesion on the MRI that was suspicious for renal cell carcinoma, and he noted that while the tumor stained positive for "CAIX and RCC, there is a question of the specificity of these tests for non-renal tumors." Dr. Chamie did not recommend a change in treatment, and felt that an MRI in one year was a good idea to see if there were other suspicious lesions.

A PET/CT scan was done on April 14, 2015. There was a left-sided organized pleural effusion, increased FDG avidity and nodularity in the medial pleura and costophrenic sulcus with evidence of progressive disease. There was left paravertebral nodularity at the level of the aortic arch. There was increased SUV activity in the posterior hemithorax, and in the left lateral costophrenic sulcus. There was worsening of the left-sided mesothelioma. Ms. Jackson returned to Dr. Cameron on April 22, 2015. She had no new symptoms. Dr. Cameron wanted additional testing prior to making a further decision regarding potential treatment, including a stress echocardiogram and MRI of the chest/diaphragm for function of the left side. An echocardiogram was done on May 1, 2015 and showed a normal left ventricular size and normal function with an estimated left ventricular ejection fraction of 60-65%. There was grade 1 diastolic dysfunction, and borderline pulmonary hypertension.

A chest MRI on May 28, 2015 showed worsening of the left-sided mesothelioma. The left diaphragm was involved but there was no evidence of extension across the left hemidiaphragm or visualized intraperitoneal nodularity. There was limited excursion of the posterior left hemidiaphragm during maximum breathing. Ms. Jackson went to see Dr. Alina Katsman, a primary care physician, on June 4, 2015. Her hypertension and diabetes were under control. Dr. Katsman noted that surgery was planned for June 29th with Dr. Cameron. Dr. Katsman saw Ms. Jackson on June 15, 2015. She had a sore throat, increased cough and thick mucus. Dr. Katsman treated her for atypical pneumonia with antibiotics (azithromycin) and Albuterol as needed. Ms. Jackson returned to Dr. Katsman one week later; she was feeling better although she still had some mucus production.

Ms. Jackson was admitted to UCLA on June 28, 2015 for a left thoracotomy, left pleurectomy and decortication. Dr. Cameron performed the surgery on June 29, 2015. Dr. Cameron found that the left pleural tumor was particularly adherent to the area just posterior to the aortic arch, to the aortic arch, and the posterior chest wall. It was also adherent to the lung hilum, the pericardium as well as to the diaphragm. There were no endobronchial lesions noted but there was external compression of the bronchial tree on the left side. Dr. Cameron then performed betadine washes of the chest cavity for a total of 45 minutes. During surgery, Ms. Jackson lost approximately 4 liters of blood and received 12 units of packed red blood cells, three units of fresh frozen plasma, one cryoprecipitate pack and one platelet pack. The pathology showed malignant biphasic mesothelioma. There were 4 of 8 lymph nodes positive for metastases, and the pericardial fat and pericardial lymph node was positive for mesothelioma. There was evidence of invasion into the chest wall. Ms. Jackson's tumor was staged T3N2M0.

She received blood transfusions after surgery as well as blood pressure support with phenylephrine and albumin. A small to moderate left sided pneumothorax developed after surgery but improved. She then developed hypotension on June 30th, with a systolic blood pressure in the 70s and elevated heart rate. An echocardiogram showed normal wall motion, no effusions or sign of tamponade, a normal right ventricle and a small collapsible inferior vena cava. She had an elevated troponin level and diffuse ST elevation and PR depressions noted on her electrocardiogram, which were consistent with pericarditis. Dr. Suh, the cardiologist, recommended colchicine for one month for the pericarditis and to prevent post-operative atrial fibrillation. They also recommended a beta blocker if she remained tachycardic, rather than the diltiazem since she was hypotensive. Ms. Jackson continued to have episodes of hypotension in the days after surgery and needed to have fluid boluses, as well as pain. She developed a pleural effusion on the right side and underwent an ultrasound-guided thoracentesis of the right chest on July 5, 2015. She had pain on the right side, which Ms. Jackson attributed to the chest tube. A CT angiogram was ordered to ensure that she didn't have a pulmonary embolism; no pulmonary embolism was seen. Small bilateral pneumothoraces and bilateral pleural effusions were noted, and a moderate pericardial effusion.

She had three chest tubes removed on July 7th, and one remained until July 10th. Ms. Jackson was also having difficulty eating, and had severe constipation, abdominal bloating, frequent burping and pain in her right mid-upper abdomen after drinking fluid. Gastroenterology consulted and felt it might be related to constipation or a mild ileus. If her epigastric pain did not resolve, then an upper endoscopy would be scheduled. The epigastric pain worsened, and Ms. Jackson had an esophagoscopy on July 12, 2015. Upon placement of the endoscopy, there was a white-yellow exudate circumferentially covering the upper esophagus and small areas that appeared bluish-purple in color. There was large amount of food present in her esophagus, with the surrounding esophagus appearing erythematous. Dr. Lewis, the gastroenterologist, was unable to safely remove the food bolus and was concerned around the inflammation and that there might be a stricture.. She was then emergently intubated to prevent aspiration, as well as intra-procedure hypotension and concomitant esophageal obstruction, and Ms. Jackson was transferred to

the intensive care unit and treated for respiratory failure and hypotension. A chest x-ray and neck films were ordered to ensure that there was no pneumomediastinum.

Ms. Jackson was then transferred to the operating room, where Dr. Cameron performed a bronchoscopy and flexible esophagoscopy, and removed the food in the distal esophagus and proximal stomach. Multiple undigested medication pills were also removed. There was no evidence of ischemia in the esophageal wall or obstruction at the gastroesophageal junction. Ms. Jackson developed atrial fibrillation post-operatively, and was treated with Amiodarone.

Ms. Jackson was extubated on July 14th, and continued to have the nasogastric tube that had been placed at the time of the second surgery. She had a swallowing study to see if she was at risk for further aspiration. She had an abnormal swallowing study and there was laryngeal penetration after the swallow. She had an edematous larynx as well. An esophagram barium swallow on July 15th showed delayed esophageal emptying with tapered narrowing of the distal esophagus and thickened mucosal folds o the distal esophagus. She was placed on thick pureed foods, and the nasogastric tube was discontinued on July 16th. She remained in and out of atrial fibrillation. An esophagram showed contrast traveling through the esophagus to the stomach and into the duodenum. Ms. Jackson was able to cough well enough to clear thin liquids. She remained in atrial fibrillation, and was on subcutaneous Lovenox along with Digoxin. A CT of the bran and CT brain angiogram and CT neck angiogram were performed on July 19th. There was no evidence for an acute infarct, intracranial hemorrhage or mass effect. There was no significant stenosis or occlusion involving the intracranial or cervical vasculature. There was a moderate right pleural effusion. There was partially visualized atelectasis versus consolidation in the left upper lobe. She was discharged home on July 21, 2015, and was to have home health services.

Dr. Katsman saw Ms. Jackson on July 31, 2015. She was swallowing thick liquids only and remained very constipated. A chest x-ray on July 31, 2015 showed a small left apical pneumothorax that was decreased. There were small bilateral pleural effusions, left greater than right. An abdominal x-ray showed a non-obstructive abdominal bowel gas pattern with a moderate stool burden in the descending/rectosigmoid colon. She tested negative for Anti-Hu antibodies (an antibody often present in those with paraneoplastic syndrome). Ms. Jackson was evaluated by Dr. Maggie Ham on August 7, 2015. Ms. Jackson was having difficulty getting enough calories on a thick liquid diet and had constant nausea. She had severe constipation. Dr. Ham noted that the persistent dysphagia could be related to an inflammatory process in the distal esophagus or malignancy. She recommended a repeat endoscopy, which Ms. Jackson and her family were reluctant to undertake given the complications surrounding the last endoscopy. She ordered a proton pump inhibitor as well as a barium swallow and nutrition consult. Dr. James Lee from cardiology saw Ms. Jackson on August 17, 2015. She was having shortness of breath and dyspnea and was on one liter of oxygen. Dr. Lee planned a repeat echocardiogram because of the possibility of a pericardial effusion noted on prior echocardiograms, and also felt that she needed anti-coagulation for the atrial fibrillation. He switched her to Apixaban. A CT scan of the abdomen and pelvis was done on August

13, 2015. There were indeterminate enhancing lesions of the liver and left kidney. A small enhancing nodule was seen adjacent to the left kidney that was hyperenhancing when compared to the spleen, and could represent a small splenule, but a peritoneal nodule could not be excluded. Bilateral pleural effusions with scattered reticular markings of the left lower lobe were noted. Gallstones were present.

Dr. Percy Lee, a radiation oncologist, saw Ms. Jackson on August 20, 2015. Ms. Jackson reported feeling weak and has been essentially bed bound after her long hospital stay. She was short of breath with very minimal exertion, and had lost over thirty pounds in the prior few months. She was still on a pureed diet due to dysphagia, and had more sputum production. Dr. Lee did not feel that Ms. Jackson was a good candidate for adjuvant chemotherapy to the chest wall due to her poor functional status. An echocardiogram was done on August 24, 2015. There was normal left ventricular size with borderline left ventricular septal hypertrophy. The left ventricular ejection fraction was about 55-60% and grade I diastolic dysfunction was present. There was a trace pericardial effusion. There was mild pulmonary hypertension.

Ms. Jackson went into hospice care [records not available for my review]. She died on October 28, 2015. She was 83 years old.

Past Medical History: Ms. Jackson had a history of a right breast lumpectomy in 2001 for ductal carcinoma in situ, followed by radiation to the right breast (4600 cGy to the right breast with a 1600 cGy boost to the right breast biopsy cavity). She had a left knee replacement in 2013 and a hysterectomy in 1975. She has a history of hypertension and diabetes. She had a stroke in 2011 that affected her right eye.

Cigarette Smoking History: Ms. Jackson never smoked cigarettes.

Occupational and Environmental History: Ms. Jackson worked as an elementary school teacher from 1954-1990. She worked at the Edmonds Elementary School, the HD Cooke Elementary School, the Powell Elementary School and Weatherless Elementary School. In her deposition testimony she did not recall any ceiling pipes with degrading insulation.

Ms. Jackson used Cashmere Bouquet talcum powder "before [she] was 12 and 13 years old", and also noted using powder while she was younger. Thus, she used Cashmere Bouquet starting in the 1940s and continuing for decades. She applied Cashmere Bouquet under her arms after bathing, and also put it in her shoes. She also stated that she used the powder on a daily basis even when she didn't "go into the tub." She shook the powder out of the can, and applied it to her body. Ms. Jackson used Cashmere Bouquet on a daily basis until she retired, when she noted that it was no longer available. She also noted that her mother used Cashmere Bouquet powder. They lived in a small apartment and shared one bathroom.

Conclusion: Ms. Jackson suffered and died from malignant mesothelioma of the left chest as a result of her exposure to asbestos from cosmetic talc. She underwent surgery

and had significant complications requiring emergent follow-up surgery. Eventually, she succumbed from the mesothelioma.

Based on the information available, it is my opinion, to a reasonable degree of medical certainty that Ms. Jackson's exposure to asbestos-containing talcum powder led to the development of her mesothelioma. She began using Cashmere Bouquet in the 1940s and continued to use it daily for decades.

The methodology and basis for my opinions follows standard methods of the medical and scientific community. Asbestos is the most well known cause of mesothelioma, and the causation of mesothelioma has been established by the quantitative history of exposure to asbestos. Thousands of individuals, from myriad professions and exposure situations have developed mesothelioma as a result of either direct or indirect exposure to asbestos. The reliance on the history of exposure to asbestos was used by seminal studies by Newhouse, Wagner and Selikoff in the 1960s, who attributed mesothelioma to asbestos exposure based solely on the history of exposure. The increased risks for mesothelioma exist for individuals who both worked directly with asbestos products and for those who worked adjacent to or in the vicinity of others who were using asbestos products, which is known as "bystander" exposure.

Asbestos and Malignant Mesothelioma General Opinions: Occupational Medicine is the field of medicine that deals with exposures to substances, toxins, conditions and agents in the workplace that are associated with increased risks of diseases. It exists as a subspecialty of Preventive Medicine that deals with identifying ways to prevent people from becoming ill. This includes identifying the sources, agents or catalysts that increase the likelihood of someone developing a disease, illness, or detrimental condition, and educating people on how to eliminate, avoid, and/or mitigate those risks. To put it simply, Occupational Medicine and Preventive Medicine involves searching for and identifying causes of diseases. This knowledge is important for those who are already ill: elimination of the catalysts can eliminate or mitigate the illness. It is also important from a public health point of view: to a large extent, the higher purpose of Occupational Medicine and Preventive Medicine is to educate and warn the public on how to eliminate, avoid, or mitigate the risks of diseases at the workplace, and to provide guidance to governments and businesses on appropriate regulations and standards concerning workplace health and safety.

One of the essential tasks of a physician of Occupational Medicine, when dealing with an individual patient, is the taking of a proper occupational history. Standard medical histories usually involve the patient explaining their reason for seeking medical attention; a listing of current symptoms, conditions, allergies, medications and other relevant medical problems; and providing some family and social history. Occasionally, a standard medical history may-but doesn't always-include identifying the patient's occupation.

A full occupational history, on the other hand, will go into details of a patient's entire work history, including details concerning their tasks and duties and their working

conditions and environment. The history will also routinely make inquiries into the patient's home or hobbies. It would also reveal what kinds of substances or agents the patient was exposed to in his or her working environment that might have occurred decades earlier. It remains the standard tool for determining exposure and has not be supplanted by quantitative measurements, which are rarely obtained, and would not, unless continuously performed on an individual (which is not feasible), fully address all exposures an individual might have had. At times, it is not possible to directly obtain an occupational history from an individual, and information concerning work and environmental experiences contained in deposition transcripts by plaintiffs, co-workers and family members can provide detailed information of that type that can be elicited from an occupational physician-obtained history.

The hallmark of occupational medicine is to connect an exposure to a hazardous substance to a disease, and identify whether there is a causal relationship. This is a critical differentiation in the field of occupational medicine; not only do we treat patients for disease, but we emphasis what hazardous substance might be causing the disease. In occupational medicine training, there are core areas of training, including epidemiology, biostatistics, toxicology, and industrial hygiene.

Asbestos and Disease: Asbestos is a naturally occurring mineral that has been used commercially for a variety of purposes for over 100 years. Asbestos is mined in the form of microscopic fibers released from the surrounding earth. Asbestos was extremely useful from an industrial perspective: it is highly resistant to heat and therefore serves as an excellent insulator and friction surface. It is also very durable, and as a fiber it can be molded into shapes and products that serve a variety of functions. However, asbestos is also highly toxic and carcinogenic when the fibers are inhaled or ingested.

While there are many "fiber types" of asbestos, as well as different sizes of the fibers, there exists consensus among scientists that exposure to *any* asbestos fiber type or size increases the likelihood of lung cancer, mesothelioma, as well as nonmalignant lung and pleural disorders. Asbestos fibers are generally divided into two categories: amphiboles and serpentine (or chrysotile). There are several varieties of amphiboles, including both commercial and non-commercial types. The three major asbestos types used in industry have been chrysotile, amosite and crocidolite. Of these three fiber types, over 95% of all asbestos used in the United States has been chrysotile. Much of the chrysotile asbestos that was used in the US was mined in Canada, where there was contamination with small amounts of tremolite, another type of amphibole asbestos. The mainstream scientific community has also long recognized, and continues to recognize today, that there is no "safe" level of exposure to asbestos regardless of fiber type or size. This position is shared by numerous United States government agencies, including the Occupational Safety and Health Administration ("OSHA", which has regulatory authority over workplaces), the Environmental Protection Agency ("EPA" which has regulatory authority over non-occupational settings), the National Institute for Occupational Safety and Health ("NIOSH", which is responsible for conducting research and making recommendations for the prevention of work-related injuries and illnesses), the World Trade Organization ("WTO"), and the national academies of science of every major

industrialized nation. The World Health Organization recently reviewed the existing literature and concluded (in 2014) that all fiber types are capable of causing asbestos related disease, including mesothelioma, and reiterated the statement that there is no safe level for exposure to asbestos.

Due to the ubiquitous use of asbestos and its presence in naturally occurring formations, there is asbestos in the ambient air in the United States, albeit at minute levels. The ambient air concentration, or "background level" has been reported to range from 0.0005 f/cc in urban areas, to 0.00005 f/cc in rural regions. These levels are thousands of times less than the current OSHA permissible exposure level of 0.1 f/cc. While it is theoretically possible to develop mesothelioma from ambient air concentrations, it has not been proven to occur at levels at or below ambient air concentrations. Given that there is no truly "unexposed" population, it would be impossible to reasonably perform such a study to determine if this were the case.

State of the Art:

In 1898 Montague Murray described interstitial fibrosis in an individual exposed to asbestos. Pancoast described radiographic changes of interstitial fibrosis in asbestos workers in 1917. Cooke described two cases of asbestosis in the 1920s, and actually used the term "asbestosis" to describe the interstitial fibrosis among asbestos workers, and also noted pleural plaques (fibrosis) in these workers.

In 1930 Merewether and Price, in their *Report on the Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry*, noted that inhaling dust containing asbestos fibers could lead to disabling and fatal lung disease. They studies asbestos workers in the textile mills in Great Britain, and noted that asbestosis could occur in large numbers of exposed individuals. Moreover, they found that the textile workers with the highest exposures had more asbestosis than workers in areas where asbestos exposure was lower. Merewether and Price noted that asbestos was a potential hazard to health in any industry where dry asbestos products were abraded or otherwise manipulated to generate dust, such as thermal insulating. They recommended warning, education and training of all those individuals who were exposed to asbestos.

Lynch and Smith noted a case of lung cancer in an asbestos worker from South Carolina in 1935. Textbooks in the 1930s, such as A.J. Lanza's textbook on dust disease, included asbestosis as a disease of concern. In 1943, the first case of mesothelioma was associated with asbestos exposure was published by Wedler in Germany. Also in 1943, Hueper from the United States Publich Health Service stated that he believed asbestos caused lung cancer. He published an editorial stating this association in the Journal of the American Medical Association in 1949.

In 1955, Doll published a seminal article that described the increased risk of lung cancer among asbestos exposed workers. By the time of Doll's epidemiology study, there had been over 60 cases of asbestos-related lung cancer published in the literature. In

1960, Wagner et.al. published a study of 33 cases of malignant mesothelioma among individuals who were exposed to asbestos in and around the crocidolite mines in South Africa. Not only were miners developing disease, but family members, individuals on the wagon routes in which the asbestos was carried and people who had played with mine tailings as children developed mesothelioma. In the early 1960s numerous studies in several countries, under different exposure scenarios, were published that showed mesothelioma in association with asbestos exposure. In fact by the end of 1964, over 700 scientific articles had been published that showed the adverse health effects of asbestos.

The Development of Diseases: When asbestos is inhaled, some proportion of the fibers can be deposited upon any component of the respiratory tract, including the nose, pharynx, conducting airways and the alveolar or gas exchanging regions of the lung. Fibers that land initially on the airways and above are cleared rapidly from the lung. The primary defense mechanism that mediated this clearance is known as the mucociliary escalator. The escalator is comprised of collated and mucous producing epithelial cells that propel inhaled fibers up to the mouth where they can be swallowed or expectorated. These epithelial lining cells are the "target cells" for cancers. Fibers that evade the mucociliary escalator can penetrate into the lower airways and lung tissue, where they can be transported through the body. Amphibole fibers tend to clear from the lung less rapidly than chrysotile fibers. Asbestos is cleared through the pulmonary lymphatics to lymph nodes and to the pleura, the target organ for pleural mesothelioma. Of the different fiber types, Suzuki, Sebastien and LeBouffant have all shown that chrysotile fibers preferentially translocate to the pleural space.

Asbestosis: The fibers that are inhaled and deposited past the escalator can cause asbestosis. These fibers deposit initially on the Type 1 and Type 2 alveolar epithelial cells. On the epithelial surfaces, some asbestos fibers activate the 5^{th} complement which attracts inflammatory cells, including foreign particles, like asbestos, from the lung. About 20% of the fibers deposited on the alveolar surfaces are enveloped by the Type 1 cells and are translocated to the underlying connective tissue (interstitial) compartment. There, the fibers can interact with interstitial fibroblasts, myofibroblasts and macrophages. Fibroblasts and myofibroblasts are the target cells for asbestos because these are the cells that synthesize and release the scar tissue matrix. (See Y. Suzuki & N. Kohyama, *Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues.*,19 Am J. Indus, Med. 701 (1990); Y. Suzuki & N. Kohyama, *Translocation of Inhaled Asbestos Fibers from the Lung to Other Tissues*., *19* Am J. Indus, Med. 701 (1991)). They produce scar tissue when the epithelial cells are injured and when the macrophages are activated. Alveolar cells and macrophages release a number of protein growth factors that stimulate the fibroblasts to multiply and produce scar tissue, and the fibroblasts and myofibroblasts also synthesize a similar array of factors that induce their own cell growth and matrix production that we recognize as asbestosis. Like *all* of the asbestos-related diseases, asbestosis is dose dependent. An individual typically needs long-term occupational exposure to develop clinical asbestosis.

The scarring process described above begins as soon as inhaled fibers are deposited on the alveolar surfaces, and microscopic asbestosis is ongoing in the lungs of

afflicted individuals for many years before any clinical signs or symptoms are presented. The initial physiological symptom of asbestosis is shortness of breath. This is caused by the scar tissue which replaces normal elastic connective tissue, this producing a stiff lung that restricts the individual from taking a deep breath. Shortness of breath also results when scar tissue thickens the alveolar-capillary membrane, the barrier across which oxygen and carbon dioxide gases are exchanged.

Pleural Plaques and Fibrosis: This is scar tissue formation in an identical manner to that described above, under asbestosis. The difference is that there is little direct deposition of asbestos fibers in the pleura. While some fibers can be inhaled through the alveolar ducts and reach the pleura directly, most fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to the pleura do so by way of pulmonary lymphatic flow. The inhaled fibers that land on alveolar surfaces and reach the interstitial compartment have direct access to lymphatic fluids which flow through these regions on the way to the pleura. The lymphatic flow carries fibers to the pleura where they interact with the sub-mesothelial fibroblasts that produce a scar tissue matrix, as described above. If the scarring is in a circumscribed pattern, the scarring is called "plaque". Investigators have shown that this injury can result in a restrictive lung disease in some individuals.

Lung Cancer: These tumors caused by asbestos typically arise in cigarette smokers, although some epidemiologic studies on asbestos-exposed non-smokers show an increased risk of developing the disease. When an individual is exposed to the cancer-causing agents (carcinogens) of both cigarettes and asbestos, the risk of getting lung cancer is increased well beyond the risk presented by exposure to either agent alone or by simply adding the risks of the two carcinogens. Epidemiologists multiply the risks of the two carcinogens since there is a clear synergy in the way asbestos and cigarette smoke combine to cause lung cancer.

Cancer is the loss of control of cell growth. Every cell in the bodies of humans and animals is under strict genetic control of the rate at which a given cell replaces itself by dividing. Cancer is caused when the specific genes that control cell division and other aspects of the cell cycle develop errors or mutations. Carcinogens induce such errors, and complete carcinogens can produce the errors with no other agent required. Cigarette smoke has a number of complete carcinogens, and all of the asbestos varieties have been shown to act as complete carcinogens. Thus, as the airway epithelial cells of the mucociliary escalator are assaulted daily by cigarette smoke and asbestos fibers, a number of cells are injured, and many exhibit genetic errors through the lifespan of the individual. In those who are susceptible to developing a cancer, one of those injured cells accumulates a sufficient number of genetic errors in genes that control cell growth to finally, after decades of exposure, lose the normal growth pattern and grow into a malignant tumor. (See Frost G, Darton A, Harding AH. *The effect of smoking on the risk of lung cancer mortality for asbestos workers in Great Britain (1971-2005)* Ann Occup Hyg 55:239-24 (2011)).

Mesothelioma: This cancer occurs when a mesothelial cell of the pleural or peritoneal surfaces develops a sufficient number of genetic errors in a set of genes that control cell

growth, as described above. Cigarette smoking has no influence on the development of mesothelioma. (See N.S. Offermans, et. al., *Occupational Asbestos Exposure and Risk of Pleural Mesothelioma, Lung Cancer, and Laryngeal Cancer in the Prospective Netherland Cohort Study,* 56 J. Occupational Envt'l Med. 1 (2014); Robinson BM. *Malignant pleural mesothelioma: an epidemiological perspective,* 1 Annals Cardiothoracic Surgery 491 (2012)).

Asbestos exposure is the only known occupational and/or environmental cause of mesothelioma in North America, and all of the asbestos varieties induce the genetic errors described above and cause this cancer. The fibers that cause mesothelioma reach the pleural surfaces through the lymphatic pathways, as explained earlier, and they interact with the target cells of the mesothelial surfaces. When a sufficient number of genetic errors have accumulated in a single mesothelial cell, this cell can undergo neoplastic transformation and grow into a deadly tumor. It typically takes many decades for a sufficient number of mutations to occur in a single mesothelial cell because of the numerous effective defense mechanisms that destroy genetically defective cells, thus explaining the long latencies known for this cancer.

All of the asbestos varieties have been shown to cause genetic errors and fibers less than five microns can bind DNA and this contributes to the development of genetic damage. Short fibers have been found to accumulate in the pleural regions of the lung as well as in mesenteric lymph nodes of the peritoneal cavity. Longer fibers may be comparatively more dangerous than short fibers (on a fiber per fiber basis), but all size ranges are capable of causing and contributing to the development of mesothelioma or any of the asbestos-related diseases. Exposure to asbestos fibers of all types and lengths are toxic, and short fibers more readily reach the mesothelial target cells of the pleura. (See Y. Suzuki & S. R. Yeun, *Asbestos Fibers Contributing to the Induction of Human malignant mesothelioma.*, 982 Annals N.Y. Acad. Sci. (2002); Y. Suzuki, et al. *Short thin asbestos fibers contribute to the development of human malignant mesothelioma: pathological evidence.*, 208 Int'l. J. Hygiene Envtl. Health 201 (2005)). Fibers of all lengths can bind to DNA and cause genetic errors that are required in the causation of cancer such as mesothelioma. Fiber burden studies of mesothelioma patients show a preponderance of chrysotile asbestos within the tumor tissue. Since the target location of mesothelioma is the pleura, the lung burden of asbestos does not reflect the fact that asbestos has moved from the lung to the pleura, where it can cause the mesothelioma to develop. (See Ronald F. Dodson, *Analysis and Relevance of Asbestos Burden in Tissue, in Asbestos: Risk Assessment, Epidemiology and Health Effects.* Risk Assessment, Epidemiology and Health Effects 78 (2d, ed. 2011); M. Silverstein, et al., *Developments in Asbestos Cancer Risk Assessment.* Am J. of Indus. Med. (2009)).

Moreover, there is ample evidence to support the conclusion that exposure to all types of asbestos fibers, including those found in talc and talcum powder products can and does cause mesothelioma. This conclusion is supported by, among others, the American Conference of Governmental Industrial Hygienists, the American Thoracic Society, the Environmental Protection Agency, the International Agency for Research on Cancer, the National Toxicology Program, OSHA, the Consumer Products Safety

Commission, the World Health Organization, and the World Trade Organization. The scientific consensus that all fiber types and sized can cause mesothelioma is also reflected in the Consensus Report of the 1997 Helsinki Conference (discussed below) and publications from the American Cancer Society and the National Cancer Institute of the National Institutes of Health.

In essence, there exists a consensus among the overwhelming majority of medical and scientific professionals and organizations that asbestos fibers of any type or size can cause mesothelioma, including chrysotile fibers. (See Dodson, Ronald F. et al., *Asbestos Fiber Length as Related to Potential Pathogenicity: A Critical Review*, 44 Am J. Indus. Med. 291 (2003); D. Egilman, et al., *Exposing the "Myth" of ABC, "Anything But Chrysotile: A Critique of the Canadian Asbestos Mining Industry and McGill University Chrysotile Studies.* 44 Am J. Indus. Med. 540 (2003); David S. Egilman & Marion Billings: *Abuse of Epidemiology: Automobile Manufacturers Manufacture a Defense to Asbestos Liability*, 11 Int. J. Occupational Envtl Health 360 (2005). 11:360-371; Egilman D. *Fiber Types, Asbestos Potency, and Environmental Causation.* 15 Int. J. Occupational Envtl. Health (2009); Finkelstein, M. *Asbestos Fiber Concentrations in the Lungs of Brake Workers: Another Look*, 52 Annals Occupational Hygiene 455 (2008); M.M. Finkelstein & C. Meisenkothen, *Malignant Mesothelioma among Employees of a Connecticut Factory that Manufactured Friction Materials Using Chrysotile Asbestos.* 54 Annals Occupational Hygiene 692 (2010); P.J. Landrigan, et al., *The Hazards of Chrysotile Asbestos, a Critical Review.* 37 Indus. Health 271 (1999); W.J. Nicholson, *The Carcinogenicity of Chrysotile Asbestos-A Review.* 39 Indus. Health 57 (2001); R.A. Lemen, *Chrysotile Asbestos as a Cause of Mesothelioma: Application of the Hill Causation Model.* 10 (2) Int. J. Occupational Envtl. Health (2004); *see also* R. Lemen, *Asbestos in Brakes: Exposure and Risk of Disease.* 45 Am. J. Indus. Med 229 (2004); EPA: *Guidance For Preventing Asbestos Disease Among Auto Mechanics.* (1986); A.H. Smith & C.C. Wright, *Chrysotile Asbestos is the Main Cause of Pleural Mesothelioma.* 30 Am. J. Indus. Med. 252 (1996); U.S. Dept. of Labor: *Working Safely with Asbestos in Clutch and Brake Linings.* (posting); U.S. Dept. of Labor, OSHA Directorate of Science, Technology and Medicine, Office of Science and Technology Assessment. *Asbestos-Automotive Brake and Clutch Repair Work*; World Health Organization, *Environmental Health Criteria 203: Chrysotile Asbestos.* International Programme on Chemical Safety (1998 Geneva)).

Asbestos fibers are very small; so small, in fact, that millions of fibers could fill the air in a room without anyone being able to perceive it with the naked eye. The fibers are odorless, cannot be seen with the naked eye, and are aerodynamic. Consequently, someone can inhale asbestos fibers without even being aware of it. The fibers are also small enough to pass through the normal respiratory defense mechanisms that the human body uses to keep out toxins and debris.

The Scientific community has even concluded that small amount of asbestos exposure can cause cancer. The Rodelsperger study indicates that exposure to asbestos below the Occupational Safety and Health Administration (OSHA) Permissible Exposure Level (PEL) of 0.1 fibers per cubic centimeter can cause disease. However, visible

asbestos-laden dust that is released into the air from the manipulation of gaskets or packing, or that is reintroduced into the respirable zone from the process of sweeping the floor, is between 2.0 and 10.0 fibers per cubic centimeter. These levels far exceed the OSHA PEL. Some of these levels even exceed the OSHA PEL issued in 1972.

Government agencies and international organizations universally recognize asbestos as a carcinogen in low levels. These agencies include the International Agency for Research on Cancer, Environmental Protection Agency, OSHA, National Institute for Occupational Safety and Health, and World Health Organization. The inhalation of asbestos fibers also does not trigger any immediate physiological reactions: the victim doesn't experience any immediate irritation, asthmatic problems, or allergic reactions. Moreover, the latency, or development period, for mesothelioma is very long: the minimum latency period is usually considered to be around 10 years with a maximal latency period well over 60 years after the last exposure. Consequently, it could be decades before someone is aware that he or she was exposed to asbestos, or it might have occurred so remotely that they do not realize they had asbestos exposure. Moreover, they may not realize that a product they used contained asbestos and thus are unaware they had exposure.

The Helsinki Criteria for Attribution: In January 1997, a conference called "Asbestos, Asbestosis and Cancer" was held in Helsinki, Finland. The conference was convened to establish criteria for diagnosis and attribution of disorders of the lungs and pleura, including mesothelioma. This was a multidisciplinary group of internationally recognized experts, consisting of pathologists, radiologists, occupational and pulmonary physicians, epidemiologists, toxicologists, industrial hygienists, and clinical and laboratory scientists specializing in tissue fiber analysis. Collectively, the members had published over 1,000 articles on asbestos and associated disorders. The conclusions of the conference were developed into a peer-reviewed Consensus Report that established the "Helsinki Criterion". Among the conclusions of the Helsinki Criterion are:

a. That, in general, reliable work histories provide the most practical and useful measures of occupational asbestos exposure; and
b. That even in the absence of other independent evidence of disease (e.g. lung fiber counts exceeding the background range for the lab in question; the presence of radiographic or pathological evidence of asbestos-related tissue injury; histopathologic evidence of abnormal asbestos content), a history of significant occupational, domestic or environmental exposure to asbestos will suffice for attribution of the disease with asbestos exposure.

Moreover, with reference to determining an occupational etiology of mesothelioma, the Helsinki Criterion Consensus Report concluded that:

a. The great majority of mesotheliomas are due to asbestos exposure;
b. Mesothelioma can occur in cases with low asbestos exposures. However, very low background environmental exposures carry only an extremely low risk;

c. About 80% of mesothelioma patients have had some sort of occupational exposure to asbestos (necessitating a carefully obtained and detailed occupational history for proper diagnosis);
d. An occupational history of brief or low-level exposure should be considered sufficient for mesothelioma to be designated as occupationally related;
e. A minimum of 10 years from the first exposure is required to attribute mesothelioma to asbestos exposure (though in most cases, the latency interval is longer);
f. Smoking has no influence on the risk of mesothelioma.

The conclusions of the Helsinki Criterion have since been adopted by, and form the general consensus of, the medical community's positions vis-à-vis mesothelioma and asbestos. (See *Consensus Report, Asbestos, asbestosis and cancer: the Helsinki criteria for diagnosis and attribution,* 23 Scandinavian J. Work Environ Health 311 (1997)). And, given the fact that about 80% of patients with mesothelioma have had some sort of occupational exposure to asbestos,[1] asbestos exposure in the workplace is a prime focus of Occupational Medicine when dealing with mesothelioma patients.

Mesothelioma is a dose responsive disease: It is my opinion that Mesothelioma and asbestos related lung cancer are dose responsive diseases in which more substantial exposures directly increases the risk for the development of these cancers. This linear dose-response relationship presented in *Asbestiform Fibers: Non-occupational Health Risks,* published by the National Research Council National Academy of Sciences in 1984, discussed herein, is neither new nor novel and generally accepted in the medical and scientific communities. As per the aforementioned Helsinki criteria, the first question usually asked of a patient diagnosed with mesothelioma, concerns how, when, and where the patient was exposed to asbestos. (*See Consensus Report, Asbestos asbestosis and cancer: The Helsinki criteria for diagnosis and attribution.* 23 Scandinavian J. Work Environ Health 311 (1997)). Because of the proven association between asbestos fibers and mesothelioma, proof of significant exposure to asbestos dust is considered to be proof of specific causation. (*See* P. Boffetta, et al., *Health Effects of Asbestos Exposure in Humans: A Quantitative Assessment.* 89 (6) Medicina Del Lavoro, 471 (1998). This causal relationship between exposure to asbestos dust and the development of mesothelioma is so firmly established in the scientific literature that it is accepted as a scientific "fact".

Malignant mesothelioma is, in general, a dose response disease where each and every significant exposure to asbestos-containing dust has been shown to contribute to cause diffuse malignant mesothelioma including pleural mesothelioma (*See also* Newman, et al., *Malignant Mesothelioma Register 1987-1999.* 74 Int'l Arch Env. Health 383 (2001), (concluding that "higher cumulative asbestos-fiber dose leads to the earlier development of mesothelioma)). As each exposure to asbestos contributes to the total amount of asbestos that is inhaled, and, in doing so, reduces the necessary period for

[1] The remaining 20% of mesothelioma patient likely had asbestos exposures that were para-occupational or are simply unidentified.

asbestos disease to develop. Therefore, each non-trivial exposure to asbestos should be considered a substantial contributing factor in the development of the malignant mesothelioma or lung cancer.

Exposure to Asbestos contaminated talc and disease

Asbestos fibers have been reported in cosmetic talcum powder for decades, in company documents, the media, FDA communications, trade organization documents and the published medical and scientific literature. Cosmetic talc has been analyzed by researchers in various countries, and has routinely been shown to be contaminated with asbestos. Exposure to asbestos contaminated talc has been shown, Cralley, et.al., in 1968 identified the association of cosmetic talc and its potential for causing asbestos related diseases, such as mesothelioma. Case reports identified asbestos from talc as the sole source of exposure in two individuals who developed mesothelioma (see Andrion & Fujiwara). In 1976 Rohl and Langer tested 20 consumer products that had been labeled as talc or talcum powder, including body powders. Of the 20 products that were tested, ten were found to contain tremolite and anthophyllite, principally asbestiform. Of note, the product that had the highest asbestos content in the Rohl and Langer study was the same product later tested by Gordon, et.al. This product was in fact, Cashmere Bouquet.

A recent paper by Gordon, et.al., Asbestos in Commercial Cosmetic Talcum Powder as a Cause of Mesothelioma in Women, evaluated the mineralogical constituents of Cashmere Bouquet and it's ability to release asbestos fibers into the breathing zone of the direct user and bystanders. In their paper, Gordon et.al. noted that the talc that was used in Cashmere Bouquet was derived from three distinct regions, where anthophyllite and tremolite asbestos were found. Gordon et.al. measured 18 million anthophyllite asbestos fibers per gram in the talcum powder. Air measurements were done by both phase contrast microscopy (PCM) and transmission electron microscopy (TEM), and significant levels of asbestos fibers were noted (anthophyllite, tremolite and some chrysotile) in the breathing zone of the individual applying the powder as well as a bystander. Results taken from the experiment in the paper show that personal measurements from the shaker container test showed a measurement by PCM of 4.8 f/cc, with an actual asbestos fiber measurement of 1.8 f/cc. Bystander measurements showed a lower, but still significant exposure of 1.35 f/cc by PCM for the bystander, and 0.5 f/cc of actual asbestos fibers. Similar measurements were done with the puff application method. Personal measurements after using a puff were 23.6 f/cc and 16.5 f/cc for the user, with actual asbestos fiber measurements of 5 f/cc and 3.5 f/cc. A short term sample showed even higher measurements, of 60 f/cc with the use of a puff and actual asbestos fiber measurements of 13 f/cc. Bystander exposures to asbestos from the puff application were elevated, with a short term sample by PCM of 13.7 f/cc and 9.7 f/cc, and an actual asbestos fiber measurement of 4.9 f/cc and 3.5 f/cc. Gordon et.al. also noted that the TEM measurements were far more sensitive than x-ray diffraction detection, since there was a much lower detection limit with TEM.

In addition to looking at bulk and air samples, Gordon et.al analyzed the lung tissue and lymph node tissue of a woman who had been exposed to Cashmere Bouquet.

The authors found that there were 3150 and 4150 fibers per gram wet weight, respectively, with a detection limit of 690 fibers per gram wet weight. All fibers were 5 micrometers or greater in length, and had an aspect ratio of 20:1 or greater. The fibers were identified as anthophyllite or tremolite. In addition to the fibers counted above, there were many anthophyllite and tremolite fibers that were less than 5 micrometers in length, with a predominance of anthophyllite. In the lymph node, amphibole asbestos fibers were also noted, measuring12,738 fibers per gram wet weight (detection limit 2123 fibers per gram wet weight). Again, the fibers noted were anthophyllite and tremolite. In addition to the asbestos found in the lungs, the authors noted fibrous and platy talc and small asbestos bodies.

The issue of asbestos and talc has been studied for decades among talc miners and millers. Lung scarring was seen in miners from New York State in the 1950s, and there are elevated rates of mesothelioma and lung cancer in miners at the asbestos contaminated talc mines (see also Kleinfeld 1967 and Finkelstein 2013). Recently, mesotheliomas in Italian chrysotile miners and millers have been attributed to tremolite asbestos-containing talc near and around that geologic formation (see Ilgren 2015). The International Agency for Research on Cancer has noted that talc contaminated with asbestos is carcinogenic.

Applying an Accepted Method for Evaluating Disease Causation in an Individual

In deciding whether Ms. Jackson's mesothelioma was caused by her exposure to asbestos, I applied the methodology that was described by Welch, et.al. in her paper Asbestos Exposure Causes Mesothelioma, but Not This Asbestos Exposure: An Amicus Brief to the Michigan Supreme Court, published in 2007 in the International Journal of Occupational and Environmental Health. In this paper, she identifies four questions that should be examined in the causation of disease in the an individual:

1. Was the individual exposed to a toxic agent?
2. Does the agent cause the disease present in the individual?
3. Was the individual exposed to this substance at a level where the disease has occurred in other settings?
4. Have other competing explanations for the disease been excluded?

For question #2, there is ample literature that asbestos causes mesothelioma and no dispute in the medical literature. Ms. Jackson has no other competing explanations (#4) for the development of her left sided mesothelioma. While there is some indication that therapeutic radiation may cause mesothelioma, this is confined to the areas in which the radiation is applied; Ms. Jackson had radiation treatments after her breast cancer to her right breast region. Her mesothelioma was on the other side of the body, away from the radiation field, and it is my opinion that her radiation treatments had no effect on the subsequent left pleural mesothelioma. With respect to question #1, Ms. Jackson has an exposure to asbestos from talcum powder for decades, fulfilling this criterion. The remaining criterion, #3 is whether there is an analogous exposure scenario in which others also developed mesothelioma. As described above in the medical and scientific

literature, as well as recently published by Gordon, et.al, there are other women and men with exposure to cosmetic talcum powder products and the source talcs who then developed malignant mesothelioma.

Summary and Specific Causation in Ms. Jackson's Case

Based on the information that was provided to me, and applying both my understanding of the medical literature and the facts of this case, it is my opinion to a reasonable degree of medical certainty that the exposures to the dust from asbestos-contaminated cosmetic talc products that Ms. Jackson used for in at least four decades, starting over 60 years ago, were above normal background levels. Her exposure to asbestos-contaminated talcum powder was the cause of her mesothelioma. If she had not used asbestos-containing talcum powder, she would not have developed malignant mesothelioma.

The opinions related to Ms. Jackson's case are based on my review of the evidence of exposure in this case, the medical and scientific literature as described above regarding asbestos exposure and disease, available studies concerning fiber release, epidemiological studies of exposure to asbestos and the development of disease, and my knowledge, skill, experience, and training as a physician specializing in occupational medicine with a clinical focus on evaluating individuals with asbestos exposure.

I have attached a partial reference list as Appendix C that indicates reliance materials for this report.

Sincerely,

Jacqueline Moline, MD, MSc, FACP, FACOEM